THE SIGNIFICANCE OF TUBERCLE BACILLI IN THE FECES.

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The detection of tubercle bacilli in solid or well-formed stools of several patients whose histories were vague and in whom the clinical diagnosis was far from clear, led me to take up this subject for research. The idea was actually to see how prevalent is the occurrence of acid-fast bacilli in the feces, by studying the feces of others than those suffering from known tuberculous infection. With this end in view during the last two years, 612 cases were collected from the wards of the Philadelphia General Hospital. Together with this number there were stools of 60 cases of diagnosticated tuberculous infection, making in all 672. The stools were obtained from patients with croupous pneumonia, typhoid fever, erysipelas, diarrhœa, surgical and nervous disorders, and from individuals who were apparently healthy; in fact, from patients in all wards of the institution, no matter what the clinical diagnosis. Of these stools 137 were solid, 297 were semisolid, and 178 were fluid. The tubercle bacillus was found in 120 cases, or 19.6 per cent.; in solid stools 28 times; in semisolid stools 40 times, and in fluid stools 52 times. In the 60 cases of diagnosticated tuberculosis, the organism was demonstrable in all, no matter what the consistency of the feces.

It has been observed by some writers that acid-fast bacilli have been found in the feces of those suffering from enteric fever, and that it is quite common for them to be found in all specimens of feces even in health; that this latter assertion is absolutely erroneous will be shown by the results of the studies here recorded. The presence of the tubercle bacillus in the feces may be a process of excretion; wherefore a few experimental studies regarding the excretion of bacteria in general may be quoted.

Newmann[1] and Karlinski[2] observed Bacillus typhosus in the urine in 35 out of 112 cases examined. Wyssokowitsch[3] in 22 experiments employed fourteen bacterial species; only twice were the species introduced into the blood, and yet they were found in the content of the intestine. On each occasion there were macroscopic hemorrhages into the serosa and mucosa. He concludes that the passage of bacteria into the excreta occurs only when the blood containing them escapes through some breach of continuity in the excretory membrane due to inflammatory or mechanical injury. Dobroklouski[4] in Cornil's laboratory found that the bacillus of avian tuberculosis, when administered with food, infected guinea-pigs by penetrating the healthy intestinal mucosa.

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[1] Berl. klin. Woch, 1890, p. 229.

[2] Prager med. Woch, 1890, p. 231.

[3] Zeits. f. Hyg., 1886, vol. i, p. 3, quoted by Sherrington.

[4] Quoted by Sherrington, Jour. Path. and Bact., 1893, p. 258.

As corroborative evidence, numerous investigators have performed experiments by feeding, by subcutaneous and intravenous inoculations, and proved that the organisms pierce and reach the mucous membrane without any evident lesion being present.

Emmerich and Buchner,[5] after injecting Bacillus neapolitanus into the blood, found that the organism escaped in large numbers through the intestinal wall. In one experiment no pathological change in the intestinal wall was observed, but in almost all there was blood in the contents of the intestine and hemorrhages in the mucosa. Sherrington,[6] in concluding his article on the escape of bacteria with the secretions, says the evidence is against believing that when the transit of bacteria across the secreting membrane occurs the membrane is still normal in condition, although at the same time it may not be ruptured or pervious to red blood cells.

The fact that the escape of the bacteria tends to occur not immediately upon the introduction of them wholesale into the circulation, but in the late stages of the communicated disease, suggests that the healthy secreting membranes are not pervious to bacteria, and that only after soluble poisons produced by the infection have had time to act upon them do the membranes become pervious to the germs.

Babes[7] concludes, from experimental work upon glanders, that the bacillus can penetrate uninjured mucous membranes. At this point may be mentioned the well-known experiments of Ravenel, who, shortly after having fed animals tubercle bacilli, killed them and found the bacilli in the thoracic duct, though no lesions or abrasions of the mucous membrane of the gut were evident.

As another instance of the penetration of mucous membranes by pathogenic organisms, typhoid bacilluria may be mentioned. This condition was studied at some length by Koujojeff,[8] by Charrin and Ruffer, Ruffer, Schweiger, Blachstein, Corrado, Pernice and Scagliosi, Cornil, and others, quoted by Sherrington.[9] They have proved, by experiments upon animals with various bacteria, that it is quite common for bacteria to be found in the bile or urine after intravenous or subcutaneous inoculation or even after feeding alone.

Griffith[10] has shown that when tubercle bacilli are injected into a cow the milk may be infective without disease of the udder.

I inoculated rabbits and guinea-pigs subcutaneously with a homogenized culture of attenuated human tubercle bacilli. Upon the fourth day I detected tubercle bacilli in their feces. After seven weeks the animals were killed. The guinea-pig showed neither a local nor visceral lesion, not even intestinal lesions, while the rabbit, though no visceral lesions were found, had a caseous mass at the site of inoculation and in the right inguinal region. A second series of guinea-pigs and rabbits was inoculated in the same manner and with the same organism. I again found tubercle bacilli in their feces at the end of the fourth day. These animals were killed at the end of the sixth week and at autopsy no intestinal, visceral, or glandular lesions were present in the guinea-pig, but the rabbit showed a large, apparently caseous mass in the right inguinal region in which I was unable to find tubercle bacilli.

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[5] Arch. Hyg., vol. xi, p. 357.

[6] Jour. Path. and Bact., 1893, p. 276

[7] Comptes-rend.. Paris, 1888.

[8] Central. f. Bact. u. Parasit., 1889, vol. vi, p. 672.

[9] Loc. cit.

[10] Royal Commission on Tuberculosis, Brit. Med. Jour., 1907, ii, 211.

Examination of a number of specimens of feces from guinea-pigs and rabbits in apparent health failed to reveal any acid-fast bacilli.

In well-defined instances of pulmonary tuberculosis it is the rule to find tubercle bacilli in the feces. If a case comes under the observation of the clinician which is not at all clear, but presents a clinical picture resembling malaria, enteric fever, or acute miliary tuberculosis, the finding of tubercle bacilli in the feces will determine the diagnosis. In those suffering from chronic diarrhœa, with no other appreciable symptoms, pulmonary or otherwise, the tubercle bacillus is at fault in most cases. These cases have frequently come to autopsy, some showing intestinal ulceration and others showing no ulceration.

Instances of ascites, the exact nature of which was unknown, were diagnosticated positively as tuberculous by the finding of the tubercle bacillus in the feces. These cases were further proved by surgical procedures, that is, abdominal operation. As a well-marked intestinal tuberculous ulcer is appreciable from the appearance of the serous coat of the gut, in one case thus operated upon, no ulcers were present.

That tubercle bacilli are in the feces, irrespective of a pulmonary or an intestinal lesion is proved by the fact that I found them in cases of general glandular involvement, meningitis, hip-joint disease, and Pott's disease of the spine, the latter condition being in a boy aged five years.

In acute miliary tuberculosis, diagnosticated or not clinically, the bacillus was present in the feces in all cases. The result of these studies suggests the intestinal mode of infection in tuberculosis in general. It is my intention merely to quote a few observations supporting the theory of the intestinal infection in tuberculosis, as these studies strengthen that theory. This theory of infection is gaining ground daily and the experiments of Schroeder and Cotton,[11] of Ravenel, and of Vallee who practically confirmed Ravenel's feeding experiments by working upon calves, of von Behring, and Calmette, and Guerin's work upon goats, demonstrate fully that the intestine is by far the most common path of infection and that aërial infection is uncommon.

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[11] Bull. No. 86, Bureau of Animal Industry of Department of Agriculture.

In a number of autopsies in which the mesenteric and other glands were studied bacteriologically, it was found that over 40 per cent. showing no tuberculous lesions in any part of the body were tuberculously infective. It was also found that in all cases of active tuberculosis and in almost all cases of inactive tuberculosis, the mesenteric glands were tuberculously infective.[12] Supporting von Behring's theory that the most frequent method is through the intestinal wall, Guthrie found over 22 per cent. of infections through the intestinal tract, Heller nearly 40 per cent. of primary intestinal affections, and Still over 23 per cent.

To explain the presence of the tubercle bacillus in the feces of man is not easy. I believe that the bacillus enters the human economy through ingestion (water?), either in infancy or maturity. At first the number of bacilli is not large, and they find their way to the blood and lymph stream. During their transit some are discharged through the feces and others through the urine. The circulation of these organisms through the lymph and blood continues indefinitely, and the patient actually suffers from a toxemia which may be so severe as to set up chronic diarrhœa, or cause vague symptoms characteristic of no one disease.

As the organisms multiply in the body and as the toxemia and irritation progress, a point of least resistance is somewhere established; the tubercle bacillus lodges and sets up the disease, with its distinct pathological features. It is interesting to note that in cases of healing tuberculosis, or actually arrested or healed cases, the tubercle bacillus is rarely if ever found in the feces. This is also true of the occurrence of tubercle bacilli in the sputum. In some of these cases recorded, the sputum has been examined at least six and as many as twelve times negatively, and subsequent examination of the feces was also negative.

Passler,[13] in considering the diagnosis of pyretic conditions, as septicemia and typhoid fever, mentions the probability of acute intestinal tuberculosis being the cause of the malady, irrespective of tuberculosis of any other organ. During the evolution of intestinal tuberculosis, he suggests that the infection by pyogenic organisms will perform the same role as they do in ulcerative pulmonary tuberculosis, and asserts that when we are dealing with a marked pyretic condition, which has lasted for several weeks without any particular and definite signs to indicate typhoid fever or septicemia, we must always think of an acute intestinal tuberculosis. He concludes by saying that as an intestinal infection may occur without any manifestations clinically the feces should be examined for the presence of tubercle bacilli. He cites only 2 cases.

Wood[14] states that tubercle bacilli are found in the feces of persons suffering from tuberculosis (pulmonary), because in the majority of cases the bacilli are swallowed together with small masses of sputum. Sahli[15] mentions that in intestinal tuberculosis tubercle bacilli are found in the feces and are therefore of diagnostic importance. "The stools may, however, contain these bacilli even though there is no intestinal tuberculosis (if the patients swallow their sputum). Searching the stools has even been recommended for the diagnosis of lung tuberculosis in cases of irresponsible persons who swallow the sputum. Previous treatment with dilute potassium hydroxide or digestive enzymes is often successful and may be serviceable in the examination of mucopurulent particles of the movement which have been isolated from the mass of feces. We do not know whether, under certain conditions, decomposition will destroy tubercle bacilli in the intestine. At any rate, we cannot always demonstrate tubercle bacilli in the stools, even when there is undoubted intestinal tuberculosis. Perhaps this is on account of the dilution of the content of tubercle bacilli by the abundant particles of food. Tubercle bacilli are most readily found in the purulent or bloody pieces of diarrhœal stools. As tubercle bacilli in the feces may be due to swallowed sputum, we can diagnosticate intestinal tuberculosis if bacilli are found in the feces only, when at the same time, attacks of diarrhœa occur with pus and blood in the stool. The tubercle bacillus must be carefully distinguished from the smegma bacillus, which is said to occur at the anal orifice and might have become mixed with the feces."

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[12] Rosenberger, A Study of the Mesenteric Glands in their Relations to Tuberculosis, AMER. JOUR. MED. SCIENCES, July, 1905.

[13] Münch med. Woch., October 23, 1906.

[14] Chemical and Microscopical Diagnosis, 1905.

Lichtheim[16] says that the presence of tubercle bacilli in the stools is the exception rather than the rule in persons suffering from pulmonary tuberculosis. He further asks the question whether the presence of these organisms in the feces means intestinal ulceration or merely that the patient has swallowed them with his sputum. In control observations he showed this not to be the case, as he claimed it was only exceptional to find the bacilli, since they were very difficult to find and then only a very few were present.

In January, 1897, Shaw[17] mentions the finding of tubercle bacilli in the feces of a patient exhibiting no tuberculous lesion of the intestinal tract. The lungs, however, showed isolated tubercles and areas of bronchopneumonia.

Emerson,[18] in remarking upon the occurrence of tubercle bacilli in the feces, says that it must always be borne in mind that the organisms may be swallowed, especially in children in whom the diagnosis of pulmonary tuberculosis has been made; "but this is rather a remote possibility in the case of a careful adult."

Boston[19] recommends collecting a "small portion of the purulent or mucoid material from the feces, smear it thinly on a slide," and then stain as for tubercle bacilli in the sputum. "Tubercle bacilli when found in the feces point conclusively to the existence of tuberculous ulceration of the intestines."

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[15] Diagnostic Methods, 1905.

[16] Fortschritte d. Med., January, 1883.

[17] Jour. Amer. Med. Assoc., March 20, 1897, p. 554.

[18] Clinical Diagnosis, 1906, p. 390.

[19] Ibid., 1904, p. 380.

Simon[20] claims that when tubercle bacilli are present in the feces, it indicates intestinal ulceration, providing they are observed upon repeated examinations and there are clinical symptoms pointing to the bowels as the seat of the disease; otherwise, they may be referable to swallowed sputum.

Technique. If the feces to be examined was fluid or semisolid, a small quantity from any part of the stool was taken and spread on a slide, dried, and stained. When the feces were solid, a small amount of sterile distilled water was put upon the slide and a small mass of fecal matter added, mixed thoroughly, spread, dried, and stained. Not one of the specimens was centrifugalized.

In staining the preparation, carbol-fuchsin was applied for fifteen minutes in the cold, the excess drained off, and Pappenheim's solution poured on the preparation. This was allowed to act for two or three minutes, washed with water, and if the specimen was of a uniform blue color it was dried and examined in cedar oil. If the preparation was not uniformly blue, Pappenheim's solution was applied and reapplied until the smear was blue. By observing this technique carefully, no mistake can happen regarding the diagnosis of the tubercle bacillus, as this organism and spores of other bacilli are the only bodies retaining the carbol-fuchsin stain. All other bacteria and cellular elements are stained blue. Great care must be taken lest some artefact be mistaken for the tubercle bacillus, such as a minute scratch in the glass, a small crystal, or the periphery of a cell. The organisms, as a rule, are comparatively few, in cases not plainly diagnosticated as tuberculous, but in well-marked cases of pulmonary or intestinal tuberculosis they are comparatively abundant. The finding of the tubercle bacillus in a spread is not always easy of accomplishment; it has frequently taken me at least an hour and sometimes as long as two hours to find three or four bacilli.

Direct searching through solid stools is less promising than in fluid stools. Nevertheless, tubercle bacilli may quite frequently be demonstrated in solid movements, if, as Hamberger[21] recommends, we mix a piece of feces the size of a pea with a few centimeters of water, then centrifuge gently to remove the coarser pieces, dilute the supernatant cloudy fluid with a double volume of alcohol, centrifuge once more, and then after drying examine the remaining precipitate which will consist almost exclusively of bacteria. Personally I have never found this procedure necessary.

Park[22] recommends searching in the feces for any purulent or mucous particles, and if none are found the larger masses are removed, the rest diluted and centrifugalized, and stained by the ordinary methods.

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[20] Clinical Diagnosis, fifth edition, 1904, p. 328.

[21] Quoted by Sahli.

[22] Pathogenic Microörganisms, 1905, p. 312.

Page[23] mixes a small mass of feces in 1.5 c.c. distilled water, adds 54 c.c. of a mixture of equal parts of alcohol and ether, centrifugalizes ten minutes, makes a smear of the sediment, fixes it to the slide with albumen, and stains as usual.

If the assertion that the tubercle bacilli found in the feces result from swallowing sputum or the presence of intestinal tuberculosis is true, the examination of the feces is useless, as no further knowledge is gained.

It is a well-known fact to students of tuberculosis that a persistent diarrhœa is present for a very long time, and yet the autopsy shows no ulcerations in any part of the intestinal canal. Therefore, it is by no means certain, if we find tubercle bacilli in the feces of those suffering from tuberculous enteritis, that ulcerative lesions are present. But, suppose that acid-fast bacilli are present in the feces of a person not suffering from any appreciable lesion of tuberculosis, what then is the significance of such a finding?

I believe from the studies made of this number of cases, both from a clinical and pathological standpoint, that if an acid-fast bacillus is present in the feces of any individual, and this organism resembles morphologically and tinctorially the tubercle bacillus, tuberculosis of some part of the body exists. I do not mean pulmonary tuberculosis, but tuberculosis of the intestines, liver, lymph nodes, peritoneum, or any viscus.

A *resume* of some of the cases that came to autopsy, with general remarks concerning some of the patients who did not succumb to the disease, is of great interest and very instructive, and is appended. In almost two-thirds of the cases I was fortunate in being able to follow the clinical findings with those at autopsy. A few of the patients were removed from the institution in a precarious condition, and although a number of them died no autopsy was permitted.

CASE I.—Clinical diagnosis, cirrhosis of liver with ascites. There was no expectoration. Three days before death delirium set in, suggesting meningitis. At autopsy miliary tuberculosis of lungs and tuberculosis of the peritoneum; no intestinal ulcerations.

CASE II.—Clinical diagnosis, chronic pleurisy. Eleven examinations of sputum were negative. At autopsy pleura was one-eighth of an inch in thickness over right lung and the same lung showed miliary tuberculosis. No intestinal ulcerations.

CASE III.—Clinical diagnosis, acute miliary tuberculosis. The sputum was examined on nine occasions with negative results. At autopsy miliary tuberculosis of lungs and spleen. No intestinal ulcerations.

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[23] Quoted by Emerson.

CASE IV.—Clinical diagnosis, acute miliary tuberculosis. At least six examinations were negative for the tubercle bacillus. At autopsy there was found acute miliary tuberculosis of all the viscera; no intestinal ulcerations.

CASE V.—Clinical diagnosis, pleurisy and tuberculosis of hip. Sputum on a number of occasions was negative for the tubercle bacillus. At autopsy there was acute miliary tuberculosis of all viscera; no intestinal ulcerations. (Three weeks after finding the tubercle bacillus in the feces, it was found in the sputum.)

CASE VI.—Clinical diagnosis, typhoid fever. No expectoration, and there were four negative Widal reactions. At autopsy six small, irregular, atypical ulcers; long axis transverse to bowel; one of these ulcers had perforated the gut. Studied bacteriologically, these ulcers showed few tubercle bacilli.

CASE VII.—Clinical diagnosis, tuberculosis of the hip. No autopsy.

CASE VIII.—Clinical diagnosis, acute miliary tuberculosis. Acute miliary tuberculosis of all viscera was found at autopsy, but no intestinal ulcerations.

CASE IX.—Croupous pneumonia and pleurisy. No tubercle bacilli found in the sputum. Crisis occurred, but there is still an irregular temperature.

CASE X.—Clinical diagnosis, chronic diarrhœa. No cough; no expectoration. No autopsy.

CASE XI.—Clinical diagnosis, chronic diarrhœa. After finding tubercle bacilli in the feces, slight impairment of resonance was noted in the right apical region.

CASE XII.—Clinical diagnosis, chronic diarrhœa and cirrhosis of the liver. No expectoration. Tuberculous ulcers were found in the gut at autopsy.

CASE XIII.—Clinical diagnosis, repeated attacks of pleurisy. No expectoration. No autopsy.

CASE XIV.—Clinical diagnosis, pleurisy with effusion. Sputum negative on four occasions for the tubercle bacillus.

CASE XV.—Clinical diagnosis, chronic diarrhœa. No expectoration. Typical tuberculous ulcers of the gut were found at autopsy.

CASE XVI.—Clinical diagnosis, erysipelas and diarrhœa. No expectoration. Slight pulmonary lesions were found; no intestinal ulcers.

CASE XVII.—Clinical diagnosis, septicemia, pelvic abscess, typhoid. Acute miliary tuberculosis of all viscera; no intestinal ulcers.

CASE XVIII.—Clinical diagnosis, malignant endocarditis. No expectoration. Acute miliary tuberculosis of all viscera; no intestinal ulcers.

CASE XIX.—Clinical diagnosis, chronic diarrhœa. At autopsy, intestinal ulcers were found.

CASE XX.—Clinical diagnosis, alcoholism. No expectoration. Recent and old lesions were present in the lungs; no intestinal ulcers.

CASE XXI.—Clinical diagnosis, tuberculous peritonitis. At autopsy tuberculous peritonitis and intestinal ulcers were found.

CASE XXII.—Clinical diagnosis, pleurisy. Tuberculosis of both pleuræ, of mesenteric glands and spleen; no pulmonary or intestinal lesions found.

CASE XXIII.—Clinical diagnosis, chronic diarrhœa. Tuberculous ulcers present.

CASE XXIV.—No clinical diagnosis made. At autopsy general miliary tuberculosis was found, but no intestinal lesions.

CASE XXV.—Clinical diagnosis, enteritis. This case was a child two years old and treated for seven months for gastro-enteritis; there was no cough, no expectoration; the cervical glands were enlarged.

CASE XXVI.—Clinical diagnosis, tuberculous peritonitis. Patient had cavities in both lungs, yet no sputum could be collected.

CASE XXVII.—Clinical diagnosis, Pott's disease. This case was a child five years old; no pulmonary lesions demonstrable. No autopsy.

CASE XXVIII.—Clinical diagnosis, typhoid fever and pneumonia. No tubercle bacilli were demonstrable in the sputum on several different occasions, and during illness two negative Widal tests resulted, and blood showed a leukocytosis of 17,000. At autopsy acute miliary tuberculosis of both lungs, liver, and spleen, with few intestinal ulcers.

CASE XXIX.—Clinical diagnosis not made. At autopsy, general miliary tuberculosis; no intestinal ulcers.

CASE XXX.—Clinical diagnosis, tuberculous peritonitis. This case was operated upon and the diagnosis confirmed.

CASE XXXI.—Clinical diagnosis, probable carcinoma of the stomach. Pernicious vomiting, chronic diarrhœa, marked cachexia, and emaciation were among the cardinal symptoms. No autopsy.

CASE XXXII.—Clinical diagnosis, typhoid fever. Child six years old, running a persistent irregular temperature; no pulmonary symptoms, and three Widal tests were negative.

CASE XXXIII.—Clinical diagnosis, secondary anemia. Besides the tubercle bacillus being found in the feces, the ova of Tricocephalus dispar and Ascaris lumbricoides were present. The malarial parasite was also seen in the blood. (As this was an immigration case, he was immediately deported.)

CASE XXXIV.—Clinical diagnosis, probable tuberculosis of the liver. Bacilli were found on two occasions in the feces. No autopsy.

CASE XXXV.—Clinical diagnosis, not made. Sputum examination negative on three occasions. Caseous tuberculosis of the bronchial and mediastinal glands; miliary tuberculosis of the kidney, liver, spleen, and intestines was found at autopsy.

CASE XXXVI.—Clinical diagnosis, chronic pleurisy. No tubercle bacilli were found in the sputum. At autopsy adhesive pleurisy on both sides was found, together with a few miliary tubercles in the lung. There was no intestinal ulceration.

CASE XXXVII.—Clinical diagnosis, erysipelas. No tubercle bacilli were demonstrable in the first three examinations of the sputum, though a fourth examination was positive. (Three days after tubercle bacilli were found in the feces, pulmonary hemorrhage took place, and the bacillus was found at this time.)

CASE XXXVIII.—Clinical diagnosis, interrupted recovery from typhoid fever. No autopsy.

CASE XXXIX.—Clinical diagnosis, typhoid fever. Acute miliary tuberculosis of a general character was found at autopsy; no intestinal ulcerations.

CASE XL.—Clinical diagnosis, chronic diarrhœa. Tuberculosis of the intestines and tuberculosis of the mesenteric glands were present at autopsy.

CASE XLI.—Clinical diagnosis, acute miliary tuberculosis. No tubercle bacilli were found in the sputum. At autopsy acute miliary tuberculosis of all the viscera was found, but no intestinal ulcers.

CASE XLII.—Clinical diagnosis, chronic diarrhœa and jaundice. *X*-rays showed gallstone. Tuberculosis of the retroperitoneal glands was observed at autopsy, but no gallstone.

CASE XLIII.—Clinical diagnosis, pneumonia, no crisis, and running an irregular temperature; no tubercle bacilli could be found in the sputum. Healed tuberculosis of the lungs, with tuberculous ulcers in the gut, were found at autopsy.

CASE XLIV.—Clinical diagnosis, typhoid fever. No expectoration. At autopsy acute miliary tuberculosis of a general character was found.

CASE XLV.—Clinical diagnosis, carcinoma of the liver. At autopsy, cancer of the lung and liver were found, together with tuberculosis of the mesenteric glands.

CASE XLVI.—Clinical diagnosis, locomotor ataxia with empyema. One liter of pus was found in the pleural cavity at autopsy. There was one positive and two negative examinations of the sputum for tubercle bacilli.

CASE XLVII.—Clinical diagnosis, general glandular enlargement. Spreads from an inguinal gland removed during life showed tubercle bacilli, and spreads from mesenteric gland removed at autopsy also contained tubercle bacilli. At autopsy there was no pulmonary tuberculosis, but a purulent peritonitis, with general enlargement of all lymphatic structures. No intestinal ulcers.

CASE XLVIII.—Clinical diagnosis, typhoid fever. At autopsy, tuberculous ulcers were observed; no pulmonary lesions.

CASE XLIX.—Clinical diagnosis, tuberculous peritonitis. Operation disclosed fibrinopurulent peritonitis. Numerous miliary tubercles on visceral and parietal layers of the peritoneum were observed, but no intestinal ulcerations could be made out.

Case L.—Clinical diagnosis, malaria. Patient had chills, fever, and sweats, occurring almost daily for ten days. Examination of the blood on several occasions failed to demonstrate the malarial parasite.

Case LI.—Clinical diagnosis, meningitis. Child, eighteen months old; no pulmonary symptoms. Tubercle bacilli were demonstrable in the spinal fluid about the same time they were found in the feces.

Case LII.—Clinical diagnosis, cervical lymphadenitis. Upon the second examination and after a very careful and prolonged search a few tubercle bacilli were found in the feces.

Case LIII.—Clinical diagnosis, cutaneous tuberculosis (lupus). Although tubercle bacilli were found in the feces, no pulmonary lesions could be made out.

Case LIV.—Clinical diagnosis, diarrhœa alternating with constipation. No autopsy.

Case LV.—Clinical diagnosis, gunshot wound of the chest penetrating the lung. On five occasions the sputum was examined for tubercle bacilli, but with negative results. At autopsy a large cavity was found which was surrounded by gangrenous tissue, and on the margin, upon histological examination, tubercles were found which showed few tubercle bacilli.

Case LVI.—Clinical diagnosis, tumor of testicle, probably tuberculous. The organ was removed and histologically presented a typical picture of tuberculosis. No pulmonary lesions were evident. It might also be mentioned that tubercle bacilli were found in sections of the organ and also in spreads before fixation.

Case LVII.—This case was one in which a large, ulcerating mass was present in each groin, following the inguinal glands. The condition had persisted for two years. Tubercle bacilli were found in the feces, though the condition had been diagnosticated as sarcomatous, specific, and tuberculous. Sections of the masses studied histologically showed typical tubercles and giant cells and tubercle bacilli.

Besides these cases just cited there were 9 cases of chronic diarrhœa, some of which, upon autopsy, showed tuberculous ulcers and others did not. In a few of these latter cases slight pulmonary involvement could be made out, while in the greater number no pulmonary lesions were demonstrable.

Conclusions. 1. No acid-fast bacillus other than the tubercle bacillus was found in the feces.

2. The presence of the tubercle bacillus in the feces means that active tuberculosis exists somewhere in the body.

3. In acute miliary tuberculosis the bacillus is always present in the feces.

4. In all cases of chronic diarrhœa and in cases of general glandular involvement, the feces should be examined for tubercle bacilli.
5. Finding tubercle bacilli in the feces does not mean intestinal ulceration in all cases.
6. In arrested or healed pulmonary tuberculosis no tubercle bacilli are found in the sputum or the feces.
7. The feces should be studied for tubercle bacilli as a part of the routine examination, especially in suggestive cases and when no expectoration can be obtained.

I wish to extend my thanks to the various resident pathologists at the Philadelphia General Hospital for their valuable coöperation.

ACUTE INTUSSUSCEPTION IN THE ADULT.

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Intussusception, although far more common in children, occurs with sufficient frequency in the adult to warrant a careful study of the causes, pathology, and results. One-third of all cases of intestinal obstruction are due to intussusception. In 1000 cases of acute intestinal obstruction reported by C. L. Gibson,[1] 187 were due to intussusception, 121 to volvulus, 354 to hernia, 42 to Meckel's diverticulum, 40 to gallstones, 34 to openings, 16 to foreign bodies, 20 to miscellaneous causes. Leichtenstern[2] found 442 cases of intussusception in a collection of 1152 of intestinal obstruction, excluding congenital obstructions, stenosis of the rectum, and the various forms of hernia. The exact proportion of cases in children and adults is about 65 to 35. It is the most common cause of intestinal obstruction in children, and one of the most uncommon in adults. Treves gives the following table of frequency by age for acute intussusception: Before the age of eleven years, 53 per cent.; between eleven and twenty years, 12 per cent.; between twenty-one and forty years, 20 per cent.; between forty-one and sixty years, 11 per cent.; beyond sixty years, 4 per cent. or less. Sixty-five per cent. of all cases occur before the age of twenty, and 53 per cent. before eleven years. Twenty-five per cent. occur during the first year of life. In chronic intussusception the percentage differs: 50 per cent. of this class of cases occurs between twenty-one and forty, and only 25 per cent. under eleven years.

[1] Annals of Surgery, 1900, p. 487.

[2] Vierteljahreschrift f. d. prakt. Heilk., 1873–74.